Cardiovascular response to haemorrhage and its modulation by concomitant injury

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A trauma victim commonly suffers a number of insults, including blood loss and injury. Each of these insults when applied individually produce their own pattern of response which interact when triggered simultaneously. The diagnosis and early treatment of haemorrhage remains a clinical problem causing significant mortality (Anderson et al. 1988), partly because the response (clinical signs) is complex and further modified by concomitant injury and drug therapy. The aim of this abstract is to describe the response to haemorrhage and discuss how it is modified by two types of injury: musculo-skeletal and blast injury to the thorax. In addition we will consider how the response may be modulated by the opioids, drugs which are commonly given to injured patients.

'Simple' haemorrhage i.e. blood loss without significant amounts of tissue damage produces a biphasic response. During the first phase there is a tachycardia and increased vascular resistance as the baroreflex strives to maintain arterial blood pressure. As the haemorrhage becomes severe a second pattern becomes apparent, characterised by a reflex bradycardia, fall in vascular resistance and hypotension (Barcroft et al. 1944; Ohnishi et al. 1997). When haemorrhage occurs on a background of musculo-skeletal injury the second phase is abolished or markedly attenuated (Little et al. 1989). Furthermore, treatment with opioid agents e.g. morphine also block the second phase of the response to haemorrhage (Evans & Ludbrook 1990; Ohnishi et al. 1997). Current studies are directed to elucidate the mechanism whereby these interactions occur. Recently, we have shown that the injury-induced blockade of the bradycardic, hypotensive response to blood loss is not mediated via the endogenous opioids. This is surprising given the effects of exogenously-administered opioids on the response to haemorrhage and the finding that the response to injury modifies other cardiovascular reflexes via the endogenous opioid system. Finally, by contrast to the effects of musculo-skeletal injury, thoracic blast injury significantly augments the second phase of the response to blood loss (Sawdon et al *in press*).

Thus, the response to blood loss is complicated by interaction with responses to other injuries and drugs that may be given to the trauma victim. A thorough understanding of these interactions and the underlying mechanisms is required to improve the diagnosis and treatment of the haemorrhaging patient.

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